with several reports of two cases each, in which the complete literature and findings of the syndrome are minutely and repetitively reviewed. By this method, the collection of enough cases adequately to study the syndrome may cover a decade or two.

The experience of every doctor in the home and at the bedside will be available to medicine only if and when the profession recognizes this hidden wealth and is willing to go to the trouble of mining it. It is inevitable that in the future a central medical registry will be established. Whether it be fostered first by local, state, or national organizations, such a means for utilizing the vast clinical resources now being thoughtlessly wasted must come. Edith Wharton, in a poem about Vesalius dying on the island of Zante, represents him as saying, "There are two ways of spreading light. To be the candle or the mirror that reflects it. There still remains to spread an answering surface to the flame that others kindle." The medical schools, the research institutions, and the large hospitals may be the candles spreading the light of medicine, but the vast body of practicing physicians is the mirror. If this reflecting surface be neglected, the flame's illuminating power will suffer.

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COMMON RECALCITRANT DERMATOSES*

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ONE feels at times, following attendance at meetings and scientific discussions, that too much stress was given to some obscure untreatable polysyllabic name, and not enough discussion on the every day management of refactory, recurring, more common skin disorders.

Chronic recurrent dermatophytosis of hands and fingers, recurrent or unimproved acne vulgaris, lichen planus, and dermatitis of external ear canals are examples of this daily group seen in clinic and office. The first two mentioned will be discussed in more detail.

The most prevalent is, I think, the chronic recurrent dermatitis of fingers and hands. The majority of these patients have made the rounds of practitioners, roentgenologists and dermatologists, who may have applied many and varied forms of local medication, and nearly all have received the limit, or near limit of superficial x-ray therapy, with added ultra-violet therapy.

There is a definite group of these recurrent cyclic type of dermatophytids, some perhaps with a superimposed contact or chemical (over-treatment) dermatitis, but distinctly not as a group, contact affairs, bacterids, or recalcitrant pustular types, described in the literature.

The usual history is the appearance, for several years, especially in summer, of a few scattered vesicles on the fingers, the first attack subsiding quickly. The second attack, a few months later may be more resistant, longer in duration, more eczematoid in nature, with clearing after two or three x-ray treatments. Each subsequent attack becomes more persistent until the skin never completely returns to normal. This group, often exposed to various local occupational irritants, sometimes patch test positive, still show recurrences, even when the irritant is removed.

Most of these patients present clinical signs of a chronic dermatophytosis of toes, feet, or toe nails, some not at the first examination, but usually all on repeated examinations, at some time during the warm summer months.

TRICOPHYTIN EXTRACT

Since 1934 I have been routinely using a stock trichophytin extract as a test and for treatment for certain resistant cases. Despite the poor and adverse reports in the literature,[1] in my hands there seems to be a distinct place for specific trichyphytin desensitization. The initial dose is small, the increase in dosage slow, the interval between doses is a week, later two to four weeks. The injections are continued, even after the injections do not show a positive reaction, after the desensitization seems complete. The local medication used was boric acid ointment or a mild tar ointment. All patients were instructed to avoid soap and water and to wear lined rubber gloves.

I have reviewed some 38 chronic, treatment-resistant cases in office practice, and found: 12 failures; 10 patients slightly improved; and 16 patients clinically cured, except for a minor spring or midsummer exacerbation of five to seven days, which was controlled easily with a single injection, and a bland ointment. Interesting, again was the observation, that except during this minor annual recurrence, all patients could follow their usual occupations, using soaps and other irritants. In this group were two medical men, four barbers, two nurses, four beauty operators, attorneys and housewives. The aggravation of the condition, with its loss of time from office and work, were of marked economic importance to all persons in this group.

The patients called improved were those not followed more than six months; but who were free of the disease at the time last seen. A total of 16 cases have been followed for two to ten years, and have been rechecked at intervals.

The 12 failures were noted in patients who had received much previous x-ray therapy, four showing changes of mild to severe radio dermatitis on fingers and hands. This observation is in agreement with others,[2] that cases previously given x-ray treatment were more slow in response to desensitization.

Here I wish to make the plea that these patients be given little or no x-ray therapy, or at most two or three (5OR) doses carefully spaced, and at the proper clinical time of subsidence of vesicles. This dosage will result in more benefit than many weekly doses; for in this small group, the percentage of minor to major skin changes due to excess x-ray dosage is far too high. Certainly we must be on guard, for these individuals, like psoriatics, soon learn that x-ray treatment controls their exacerbations faster than other forms of therapy, and they are often persistent in their demands for more treatment.

I am not asking for a revival and wholesale injection of trichophytin extract, but do feel that it is a distinct aid in selected chronic, resistant, cases, and that the immunilogic approach is perhaps the most rational.

ACNE VULGARIS

It has been estimated that approximately 80 per cent of acne vulgaris will clear and remain well, following usual standard type treatments, including drying lotions, diet, regulation, and superficial x-ray therapy.

It is the 20 per cent of unhealed or recurrent acne who present the same problems as did the first group described. These patients have also received the limit or near-limit amount of superficial x-ray therapy, vaccines, and other treatment, with indifferent results. Many are depressed or melancholic, self-conscious, stay-at-home persons.

In the past 18 months, I have treated 11 such patients,

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all females, ages 16 to 22. All had previously received three to eight months standard treatment as described above, and all clinically, were severe acne cases Two patients showed mild dryness and slight atrophy of skin, suggesting again the limit of x-ray therapy.

VITAMIN A

Three years ago, following the reports of one of our previous chairmen, I began the use of larger doses of vitamin D. My results were poor and indifferent; so I was vitamin shy, until another article by[3] on larger doses of vitamin A revived my interest.

The patients in this group were given 150,000 units of vitamin A daily for three months, together with staphylococcus toxoid (Lederle) in small weekly injections, intradermally. No local medication was given, but in weekly sessions, evacuation of pustules and comedo extractions were done in all patients. Three patients were unimproved in two months, and did not return for further treatment or observation; eight have shown complete recovery in two to six months; two patients have an occasional pustule at menstruation periods. These eight patients have been rechecked after 12 months, and still remain acne free. Two showed marked gain in weight, averaging seven pounds per month. Here, further clinical study is needed and in a much larger series of cases.

I make the plea of discontinuing x-ray therapy, unless marked improvement is seen long before the borderline dosage is approached. Perhaps many patients may not need any form of light therapy, and we may have a means of controlling acne without the hazard of actinic dermatitis.

In final summary, your chairman is desirous of stimulating more clinical reports in our meetings, with reference to management of these and other common dermatoses. This final plea is that decreased amounts of superficial x-ray therapy should be given to the average individual patient. Perhaps, in a few years, other measures may force radiation in the distant background.

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CEREBRAL VASCULAR ACCIDENTS: THEIR EFFECT ON THE STATE OF CONSCIOUSNESS*

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THE classical types of cerebral vascular accident are hemorrhage, embolism and thrombosis. The consequences are hematoma, infarct and encephalomalacia, lesions which obviously result from escape of blood out of vascular channels or from impairment of blood flow. Inasmuch as gross occlusion or rupture of cerebral vessels are seldom demonstrated in pathologic material, there is an increasing interest in functional vascular changes such as vasospasm and vasoparalysis.

Anyone who keeps himself informed of recent clinicopathologic and experimental observations will know that escape of blood into the brain or cerebrospinal fluid, or elevation of intracranial pressure short of systolic blood pressure do not necessarily cause loss of consciousness. Even the obstruction of a major cerebral artery or the rapid destruction of a sizable segment of brain tissue may cause defects in certain fields of consciousness only, rather than inevitable and total loss of consciousness. It would be interesting to discuss these anomalies in detail, but it is better to use this space in making clear what mechanisms do result in loss of consciousness, and how they operate in cases of cerebral vascular accident.

We are all acquainted with the generalization that embolism is a matter of seconds, hemorrhage of minutes and thrombosis of hours, until the appearance of paralysis and unconsciousness. It soon occurs to anyone who sees many autopsies that the clinicopathologic correlation outlined needs some revision if accurate diagnosis is to be made. The author wishes to indicate the general lines along which revision is now possible, by discussing cerebral vascular accidents from the standpoint of consciousness.

CONSCIOUSNESS

Consciousness is a complex function comprising sensation, perception, association and memory. It may be qualified as clear or clouded, intact or defective. Evidence from cases of trauma, infection, neoplasm and vascular lesions points to the upper midbrain and adjoining thalamus as the center for regulation of consciousness. Because of its location in the tentorial notch this region is vulnerable to displacement and distortion by pressure transmitted from remote as well as adjacent lesions. According to Scheinker[1] venous engorgement with edema and hemorrhage is an important factor in disorganizing the midbrain and consequent fluctuation in consciousness. The proximity of a lesion to the midbrain, and the rapidity of its expansion determine the stage at which consciousness is affected. When an extensive lesion occurs in or immediately adjacent to the midbrain, loss of consciousness occurs early, masking other clinical symptoms. A lesion affecting more remote parts of the brain results in vertigo, nausea, vomiting, headache and paresthesia or paralysis perhaps with loss of consciousness later on.

Focal lesions which do not affect the midbrain cause deficits of consciousness by interfering in greater or lesser degree with sensation, perception, association and memory in particular fields. The patient shows lack of insight into his impaired functional state at first, but in most cases he sooner or later grasps the situation.

In one common clinical type of cerebral vascular accident there is abrupt loss of consciousness which persists from a few minutes to hours or days, clearing gradually with more or less severe residual defect of cerebral function, or terminating fatally with no evident lesion other than one or more poorly circumscribed areas of softening, remote both from vital centers and from the center of consciousness in the midbrain. It has been customary to designate such episodes as expressions of arteriospasm.

Although Raynaud's discovery of local syncope and asphyxia with arteriospasm was extended by Osler to explain cases of transient paresthesia, paralysis and convulsion, the status of spasm as an etiologic factor in cerebral vascular accidents is not clear. Vasospasm is a possible cause of one type of syncope resulting from carotid sinus stimulation. It may have to do with migraine and epilepsy. With hypertension, particularly in toxemia of pregnancy, acute lead poisoning and acute glomerulonephritis transient cerebral crises with convulsion and coma, occur as symptoms of vaso spastic encephalopathy. In these conditions permanent paralysis is rare.

When in cases of advanced arteriosclerosis one ob-

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